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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/966,147	09/27/2001	Leonard G. Presta	GENENT.33CPC4C	4067

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Knobbe Martens Olson & Bear LLP
2040 Main Street
Fourteenth Floor
Irvine, CA 92614

EXAMINER

UNGAR, SUSAN NMN

ART UNIT	PAPER NUMBER
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1642

DATE MAILED: 01/15/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.
09/966,147

Applicant(s)
Presta et al

Examiner
Ungar

Art Unit
1642



-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE three MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136 (a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on Oct 31, 2003.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1, 4-14, and 23 is/are pending in the application.
- 4a) Of the above, claim(s) 8, 9, and 11-13 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1, 4-7, 10, 14, and 23 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claims _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved by the Examiner.
If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

- 13) ☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
a) ☐ All b) ☐ Some* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. _____.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
*See the attached detailed Office action for a list of the certified copies not received.
- 14) ☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).
a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892) 4) ☐ Interview Summary (PTO-413) Paper No(s). _____
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948) 5) ☐ Notice of Informal Patent Application (PTO-152)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s). _____ 6) ☐ Other: _____

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1. The Election filed October 31, 2003 in response to the Office Action of September 23, 2003 is acknowledged and has been entered. Claims 2-3, 15-22, 24-25 have been canceled, claims 1, 11-14 have been amended. Claims 1, 4-14 are currently pending in the application and Claims 8-9, 11-13 ~~and 23~~ have been withdrawn from further consideration by the examiner under 37 CFR 1.142(b) as being drawn to non-elected inventions. Claims 1, 4-7, 10, 14 and 23 as drawn specifically to a method for the treatment of a pathological condition associated with elevated neurotrophin production comprising contacting said subject with a therapeutically effective amount of an antagonistic antibody specific for human trkC are currently under prosecution.

2. Applicant's election with traverse of Group 14, claims 1, 2, 5-7, 10-14 is acknowledged. The traversal is on the ground(s) that the inventions of Group 14 and 4 should be examined together because they contain the same method steps, are closely related in their objectives and require grossly overlapping search. The argument has been considered but has not been found persuasive because the objectives are clearly different, and the literature search, particularly relevant in this art, is not coextensive. Different searches and issues are involved in the examination of each group. For these reasons the restriction requirement is deemed to be proper and is therefore made FINAL. Applicant further argues that for the same reasons the inventions of Groups 14 and 43 should be examined together and that Examiner has erroneously referred to the method characterized by the over-expression of human trkC. The argument has been considered and has been found persuasive. Examiner apologizes for the misreading of the claim and claim 23, as

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drawn to a method for the treatment of a pathological condition associated with elevated neurotrophin production comprising contacting said subject with a therapeutically effective amount of an antagonistic antibody specific for human trkC as elected for Group 14 is hereby rejoined to Group 14. Applicant further argues that SEQ ID NO:6 and SEQ ID NO:8 are splice variants, one of the other and that SEQ ID NO:8 is a truncated version of SEQ ID NO:6. The argument has been considered and SEQ ID NO:8 has been rejoined to Group 14.

3.. If applicant disagrees with any rejection set forth in this office action based on examiner's establishment of a priority date of August 5, 1994 for the instantly claimed application serial number 09/966,14, applicant is invited to submit evidence pointing to the serial number, page and line where support can be found establishing an earlier priority date for SEQ ID NOS 6 and 8.

4. Upon review and reconsideration, it has been determined that the claims of the instantly examined group includes a linking claims. The restriction requirement among the linked inventions is subject to the nonallowance of the linking claim(s), claim 1, drawn to inhibiting a biological mediated by TrkC, *in vivo*. Upon the allowance of the linking claim(s), the restriction requirement as to the linked inventions shall be withdrawn and any claim(s) depending from or otherwise including all the limitations of the allowable linking claim(s) will be entitled to examination in the instant application. Applicant(s) are advised that if any such claim(s) depending from or including all the limitations of the allowable linking claim(s) is/are presented in a continuation or divisional application, the claims of the continuation or divisional application may be subject to provisional statutory

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and/or nonstatutory double patenting rejections over the claims of the instant application. Where a restriction requirement is withdrawn, the provisions of 35 U.S.C. 121 are no longer applicable. *In re Ziegler*, 44 F.2d 1211, 1215, 170 USPQ 129, 131-32 (CCPA 1971). See also MPEP § 804.01.

Group 1. Claims 1, 2, 4-7, 10-11 are drawn to a method for inhibiting inflammatory pain, classified in Class 435, subclass 7.1.

Group 2. Claims 1, 2, 4-7, 10, 12 are drawn to a method for inhibiting tumor development, classified in Class 435, subclass 7.1.

Group 3. Claims 1, 2, 4-7, 10, 13 are drawn to a method for inhibiting cancer development, classified in Class 435, subclass 7.1.

Group 4. Claims 1, 2, 4-7, 10, 14 are drawn to a method for inhibiting neuronal sprouting, classified in Class 435, subclass 7.1.

5. The inventions are distinct, each from the other because of the following reasons:

Inventions 1-4 are materially distinct methods which differ at least in objectives, method steps, reagents and/or dosages and/or schedules used, response variables, and criteria for success.

6. Because these inventions are distinct for the reasons given above and have acquired a separate status in the art as shown by their different classification and/or recognized divergent subject matter, restriction for examination purposes as indicated is proper.

7. A telephone call was made to Ginger R. Dreger, on January 7, 2004 to request an oral election to the above restriction requirement. Ms. Dreger made an

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oral election to the above restriction requirement, a provisional election was made with traverse to prosecute the invention of Group 4, claims 1, 2, 4-7, 10, 14. Affirmation of this election must be made by applicant in responding to this Office action.

Objections

8. The specification at p. 3, discloses the following, "This is summarized in Figure 1" at lines 5-6. However, this is incorrect, it is Figure 4 that summarizes the forms of trk receptors.

9. Claims 1, 4-7, 10, 14 and 23 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. .

The claims are drawn to a method for inhibiting a biological activity mediated by human trkC receptor comprising contacting said receptor with an antagonistic antibody specific for said neurotrophin receptor, wherein said receptor comprises SEQ ID NO:8/6, wherein said biological activity is aberrant neuron spouting *in vivo*, as disclosed in the specification. The specification teaches that porcine, mouse and rat trkC have been identified as receptors which bind neurotrophic factors (p. 2, lines 30-32), wherein at least four forms of trkC (several without functional tyrosine kinase domains and two with small inserts in the tyrosine kinase domain) are known (p. 3, lines 1-6). Cells expressing TrkC bind NT-3, but not other known neurotrophic factors and various trk receptors, arising from alternate splicing events, can activate different intracellular signaling

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pathways and therefore presumably mediate different physiological functions *in vivo* (p. 3, lines 11-16). Neurotrophins have been implicated in the mediation of inflammatory pain and are overexpressed in certain types of malignancies. Accordingly, inhibitors of neurotrophin biological activity have therapeutic potential such as in pain medication and as chemotherapeutics in cancer treatment (p. 4, lines 1-10). The invention is based on the identification, cloning and sequence of naturally-occurring forms of trkC receptors from the human and the determination of their expression pattern in various tissues by Northern and *in situ* hybridization analysis. The specification identifies regions of trkC required for receptor binding and/or biological activity and discloses an immunoglobulin chimera comprising extracellular domain of trk receptors which block biological activity of the trk ligands (p. 4, lines 25-32). The specification teaches that the term "trk" with or without an affixed capital letter, i.e. "C" include "native" receptors from any animal species including full length receptors, their truncated and variant forms, such as those arising by alternate splicing and/or insertion, and naturally occurring allelic variants as well as functional derivatives of such receptors (p. 13, lines 1-7). As drawn specifically to human trkC, the term trkC polypeptide includes not only the forms cited above but also Tk domain-deleted, insertion variants of full length or truncated native human trkC and any other naturally-occurring human trkC polypeptides that might be identified in future (p. 13, lines 10-20). Functional derivative of a native polypeptide is a compound having a qualitative biological property in common with the native polypeptide (p. 14, lines 7-20). Antagonists of trkC receptor polypeptides are believed to be useful for treating kidney disorders,

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lung disorders, cardiovascular disorders, various types of tumors, aberrant sprouting in epilepsy (p. 68, lines 19-29). The specification further teaches that trkC clones were obtained from human brain libraries which did not encode a TK domain but instead showed an alternate truncated intracellular domain (p. 74, lines 19-25). The TK domain of trkC obtained in the cDNA clones contained an apparent insert of 14 amino acids between subdomains VII and VIII (p. 75, lines 5-10). One of the proposed roles for the truncated forms of the trks is to act as a dominant negative influence on signal transduction by neurotrophin in the expressing cells (p. 76, lines 16-17). This is consistent with the relative lack of efficacy of neurotrophin signaling seen in tissue from the adult brain when stimulated by neurotrophins as the ratio of truncated to non truncated forms of the trks is quite high in the adult. TrkC is differentially spliced in humans, wherein there are two forms with and without an insert of nine amino acids in the extracellular domain. The differences in binding or activity of these two forms is unknown (p. 77, lines 1-7). Various forms of human trkC, presumably due to alternate splicing, are found in the intracellular part of the molecule (p. 77, lines 1-15). It has been shown that expression of trkC, with no insert in the TK domain, confers on the expressing cells the ability to respond to NT3 with neurite outgrowth. Cells expressing trkC containing a TK insert do not respond to NT3 with neurite outgrowth (p. 77, lines 17-29). Northern analysis of trkC showed, as might be expected from the great number of potential splice variants detected while cloning trkC, a complex pattern of hybridization. TrkC is expressed abundantly in brain and there is widespread expression of trkC outside the nervous system with the greatest expression in brain, kidney, lung and

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heart and the ratio of TK containing to truncated trkC was higher in fetal compared to adult brain (p. 79, lines 5-11). TrkC was strongly expressed in ganglia from both 6 and 8 week embryos. The *in situ* hybridization analysis of expression of the members of the trk family in the human nervous system confirmed that the overall expression pattern is similar to that seen in other mammals. This should provide a foundation for further studies designed to examine the expression of the differently spliced forms of the human trks in detail in certain areas of normal and pathological tissues (p. 81).

One cannot extrapolate the teaching of the specification to the enablement of the claims because the claims as written and the invention elected are drawn to inhibition of a biological response *in vivo* wherein the biological response *in vivo* is aberrant neuron sprouting and the method comprises inhibiting the response by contacting human trkC with an antagonistic antibody that is specific for human trkC. Inherent in the method is the treatment of a disease involving aberrant neuron sprouting with a trkC antagonist antibody. However, no nexus has been established between trkC and any disease, and no nexus has been established between trkC and any population of cells wherein inhibition of trkC would inhibit said aberrant neuron sprouting. Although the specification states that antagonists of trkC receptor polypeptides are believed to be useful for treating aberrant sprouting in epilepsy (p. 68), there is no explanation as to why this statement is made. Further, although the specification clearly discloses that numerous splice variants of human trkC are known, the specification does not teach which of those splice variants would be useful in the instant method. In particular, the specification teaches that

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the term “trk” with or without an affixed capital letter, i.e. “C, include ‘native’ receptors from any animal species including full length receptors, their truncated and variant forms, such as those arising by alternate splicing and/or insertion, and naturally occurring allelic variants as well a functional derivatives of such receptors (p. 13, lines 1-7). As drawn specifically to human trkC, the term trkC polypeptide includes not only the forms cited above abut also TK domain-deleted, insertion variants of full length or truncated native human trkC and any other naturally-occurring human trkC polypeptides that might be identified in future. The specification does not teach which of these claimed human trkC’s to antagonize so that the invention will function as claimed. The specification further teaches that the various forms of trk receptors can activate different intracellular signaling pathways and therefore presumably mediate different physiological functions *in vivo*. Which one, if any, mediates aberrant neuron sprouting? Neither the specification nor any art of record teaches what the alternative signaling pathways for the receptors possessing truncated intracellular domains or for the receptors having small inserts in the tyrosine kinase domain are. Therefore, the specification fails not only to demonstrate that antagonists of trkC receptor could function in inhibiting aberrant neuron sprouting, but fails even to teach which trkC receptor would function as claimed. In particular, as drawn to epilepsy, Kim et al (Epilepsia, 2002, 43/SUPPL. 5:220-226), teaches that immunohistochemical staining was performed on surgical specimens of 20 patients with cerebral cortical dysplasia, one of the important causes of intractable epilepsies, wherein it was found that trkC was strongly expressed. The authors hypothesize, because NT-3 contributes to the differentiation

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of neuronal precursor cells, dendritic and axonal arborization, synaptic plasticity and cellular hyperexcitability, trkC **may** (emphasis added) have a critical pathogenetic role in epileptogenicity in dysplastic neurons of CD. It is pointed out that eight years post filing, those of skill in the art did not know whether trkC had a pathogenic role in epileptogenicity or whether it was involved with epileptic disease processes. Given the art recognized overexpression of non-productive trkC in adults compared with productive trkC, it is clear that the authors could not make a firm prediction as to the involvement of trkC perhaps because it would have been expected that the majority of the expressed trkC would be non-productive. Thus the effects of an antagonist on this population would have been expected to be nil. Given the above, it cannot be predicted that the invention will function as claimed with any expectation of success. Further, inherent in the "contacting" step of the claimed invention is the administration of the antagonist antibody. The specification does not provide teachings to establish effective dosages or methods of administration of antibodies specific for trkC in order to practice the invention as claimed.. In addition variables such as biological stability, half-life or clearance from the blood are important parameters in achieving successful therapy. The antibody may be inactivated *in vivo* before producing a sufficient effect, for example, by proteolytic degradation, immunological activation or due to an inherently short half life of the protein. In addition, the antibody may not otherwise reach the target because of its inability to penetrate tissues or cells where its activity is to be exerted, may be absorbed by fluids, cells and tissues where the antibody has no effect on the aberrant neuron sprouting to be treated. This is of critical

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importance given the clear teaching of the specification of the number of tissues which express the receptor and the number of tissues, including the nervous system that apparently express non-productive receptors that do not comprise a TK domain. In particular, the specification suggests that the role for the truncated forms of the trks is to act as a dominant negative influence on signal transduction by neurotrophin in the expressing cells. The effect of this upon sequestration of the antagonistic antibody cannot be predicted. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that inhibition of trkC would be useful for treatment of diseases involved with aberrant neuron sprouting or that the invention would function as claimed. For the above reasons, it appears that undue experimentation would be required to practice the claimed inventions with a reasonable expectation of success.

10. Claims 1, 4-7, 10, 14 and 23 are rejected under 35 USC 112, first paragraph, as lacking an adequate written description in the specification.

Claims 1, 4-7, 10, 14 and 23 are drawn to an *in vivo* method of inhibiting aberrant neuron sprouting by contacting said trkC receptors with an antagonist antibody. Although drawn to DNA arts, the findings in University of California v. Eli Lilly and Co., 119 F.3d 1559, 43 USPQ2d 1398 (Fed. Cir. 1997) and Enzo Biochem, Inc. V. Gen-Probe Inc. are relevant to the instant claims. The Federal Circuit addressed the application of the written description requirement to DNA-related inventions in University of California v. Eli Lilly and Co., 119 F.3d 1559,

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43 USPQ2d 1398 (Fed. Cir. 1997). The court stated that “[a] written description of an invention involving a chemical genus, like a description of a chemical species, ‘requires a precise definition, such as by structure, formula, [or] chemical name,’ of the claimed subject matter sufficient to distinguish it from other materials.” *Id.* At 1567, 43 USPQ2d at 1405. The court also stated that

a generic statement such as “vertebrate insulin cDNA” or “mammalian insulin cDNA” without more, is not an adequate written description of the genus because it does not distinguish the genus from others, except by function. It does not specifically define any of the genes that fall within its definition. It does not define any structural features commonly possessed by members of the genus that distinguish them from others. One skilled in the art therefore cannot, as one can do with a fully described genus, visualize or recognize the identity of the members of the genus. A definition by function, as we have previously indicated, does not suffice to define the genus because it is only an indication of what the gene does, rather than what it is.

Id. At 1568, 43 USPQ2d at 1406. The court concluded that “naming a type of material generally known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material.” *Id.*

Finally, the court addressed the manner by which a genus of cDNAs might be described. “A description of a genus of cDNAs may be achieved by means of a recitation of a representative number of cDNAs, defined by nucleotide sequence, falling within the scope of the genus or of a recitation of structural features common to the members of the genus, which features constitute a substantial portion of the genus.” *Id.*

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The Federal Circuit has recently clarified that a DNA molecule can be adequately described without disclosing its complete structure. See Enzo Biochem, Inc. V. Gen-Probe Inc., 296 F.3d 1316, 63 USPQ2d 1609 (Fed. Cir. 2002). The Enzo court adopted the standard that “the written description requirement can be met by ‘show[ing] that an invention is complete by disclosure of sufficiently detailed, relevant identifying characteristicsi.e., complete or partial structure, other physical and/or chemical properties, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics. ” Id. At 1324, 63 USPQ2d at 1613 (emphasis omitted, bracketed material in original).

The inventions at issue in Lilly and Enzo were DNA constructs per se, the holdings of those cases are also applicable to claims such as those at issue here. A disclosure that does not adequately describe the population of cells associated with aberrant neuron sprouting cannot adequately describe a method of inhibiting aberrant neuron sprouting.

Thus, the instant method may provide an adequate written description of said population associated with aberrant neuron sprouting wherein said sprouting is inhibitable by an antagonist antibody against trkC, per Lilly by structurally describing a representative number of such populationss or by describing “structural features common to the members of the genus, which features constitute a substantial portion of the genus.”, that is disclosing that each of those populations are associated with trkC in such a manner that antagonism of said receptor will inhibit aberrant sprouting. Alternatively, per Enzo, the specification can show that

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the claimed invention is complete “by disclosure of sufficiently detailed, relevant identifying characteristics, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics.”

In this case, the specification does not describe said aberrant sprouting in a manner that satisfies either the Lilly or Enzo standards. The specification does not provide the information regarding any disease or population of cells associated with trkC wherein the claimed antagonist antibody could inhibit aberrant neuron sprouting other than epilepsy, nor does the specification provide any physical or chemical characteristics of any disease nor any functional characteristics coupled with a known or disclosed correlation between structure and function in any disease or population of cells. Although the specification discloses epilepsy, no particular population of cells is disclosed and this does not provide a description that would satisfy the standard set out in Enzo.

The specification also fails to describe the claimed method by the test set out in Lilly. The specification simply mentions that antagonists of trkC are thought to be useful to treat aberrant neuron sprouting in epilepsy. Therefore, it necessarily fails to describe a “representative number” of such species. In addition, the specification also does not describe “structural features common to the members of the genus, which features constitute a substantial portion of the genus.”

Thus, the specification does not provide an adequate written description of the claimed invention that is required to practice the claimed invention. Since the specification fails to adequately describe the disease of population of cells to which

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the antagonist must be targeted for the method to function as claimed, it fails to describe the method.

11. If Applicant were able to overcome the rejections set forth above, Claim 23 would still be rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method wherein the condition is associated with elevated NT3, does not reasonably provide enablement for the condition is associated with elevated endogenous neurotrophin production. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make/use the invention commensurate in scope with these claims.

The claim is drawn to a condition associated with elevated neurotrophin production wherein the inhibited receptor is trkC. This includes all neurotrophins. The specification teaches that only NT-3 activates trkC.

One cannot extrapolate the teaching of the specification to the scope of the claim because no other neurotrophin, other than NT-3, was known to activate trkC at the time the invention was made. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the invention would function as claimed with any other neurotrophin with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

12. No claims allowed.

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13. ~~10.~~ Any inquiry concerning this communication or earlier communications from the examiner should be directed to Susan Ungar, PhD whose telephone number is (703) 305-2181. The examiner can normally be reached on Monday through Friday from 7:30am to 4pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Anthony Caputa, can be reached at (703) 308-3995. The fax phone number for this Art Unit is (703) 308-4242.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Effective, February 7, 1998, the Group and/or Art Unit location of your application in the PTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Group Art Unit 1642.

Susan Ungar
Primary Patent Examiner
January 12, 2004

